A LECTURE

ON

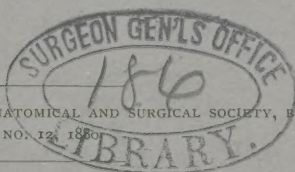# THE LOCALIZATION OF DISEASES IN THE SPINAL CORD,

DELIVERED BEFORE THE

ANATOMICAL AND SURGICAL SOCIETY OF BROOKLYN,

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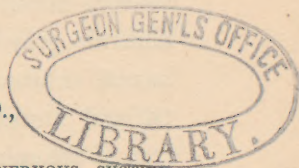
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THE LOCALIZATION OF DISEASES IN THE SPINAL CORD.

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I HAVE designed this lecture as a pendant to Dr. Gray's discourse upon the new anatomy of the spinal cord and its intra-cranial expansion. In the last fifteen years advances in the normal and pathological anatomy and the physiology of the nervous centres have progressed hand in hand, one illustrating and confirming the other.

At the beginning of this century, and from that time until some fifteen years ago, though great progress was achieved in the clinical description of spinal diseases, from Ollivier to Brown-Séquard ; and though we have acquired some knowledge of the pathological anatomy of the points involved, almost no attempt had been made to localize the lesions of various diseases in definite sections of the spinal cord. The admirable spinal localization of the present day, we owe to the accumulated labors of Cruveilhier, Türck, Goll, Charcot, Leyden and Erb.

I shall offer you to-night a brief review of this localization

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of diseases in the spinal cord, as founded upon normal anatomy, physiology and pathological anatomy. Such a study will be facilitated by admitting as practically correct a somewhat rough division of the spinal cord into two unequal regions, one including the posterior columns and the posterior gray matter, serving for the transmission of centripetal or sensory impulses, and the second, much larger, including the anterior gray horns, the anterior columns and the antero-lateral columns, serving for the transmission of motor, centrifugal impulses. The former region is the æsthesodic system of the spinal cord, the latter the kinesodic system. The accompanying wood cut illustrates the limits of these two systems, on a transverse section of the spinal cord.
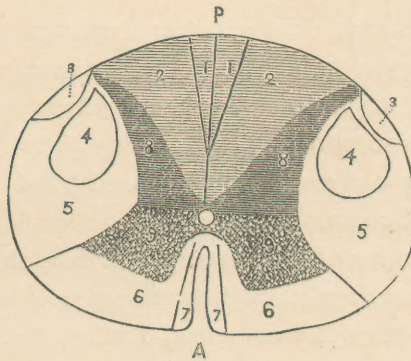

FIG. I.

DIAGRAM OF TRANSVERSE SECTION OF THE SPINAL CORD.

A. Anterior median fissure. P. Posterior median septum. 1. Columns of Goll. 2. Columns of Gerlach. 3. Direct cerebellar fasciculi. 4. Crossed pyramidal columns. 5. Lateral columns. 6. Anterior columns. 7. Direct pyramidal columns. 8. Posterior gray horns. 9. Anterior gray horns. Stippled part—Gray matter. Shaded part—Æsthesodic system. Unshaded part—Kinesodic system.

The following table embodies the principal localized lesions of the spinal cord which give rise to definite symptom-groups, and which we are able to diagnosticate:

LOCALIZED SPINAL DISEASES.

A. Systematic diseases of the spinal cord:

1. Diseases of the æsthesodic system—

    a. Sclerosis of the posterior columns (progressive locomotor ataxia).

    b. Ascending degeneration.

2. Diseases of the kinesodic system—

    a. Degeneration of anterior ganglion cells (progressive muscular atrophy).

    b. Inflammation of anterior gray horns (atrophic spinal paralysis of adults and children).

    c. Sclerosis of the lateral columns (tetanoid paraplegia).

    d. Descending degeneration,

        α.—of spinal origin;

        β.—of cerebral origin.

B. Focal affections of the spinal cord (injuries, tumors, foci of softening, myelitis transversa, clots, etc.):

    a. In lumbar enlargement.

    b. In lower dorsal region.

    c. In upper dorsal region.

    d. In lower cervical enlargement.

    e. In upper cervical region.

    f. At the cauda equina.

It will be impossible to do more this evening than to consider the first of the two great divisions—viz., that of systematic lesions.

1. Diseases of the æsthesodic system:

At the present time there is only one disease of this class —viz., posterior spinal sclerosis or progressive locomotor ataxia. The lesion consists, roughly speaking, in sclerosis of the posterior columns of the spinal cord. Since 1873 closer analysis at the hands of Charcot and Pierret has shown that the primary and essential sclerosis occupies only the external part of these columns—that which is adjacent to the posterior gray horns, and which histology teaches us is traversed by fibres of the posterior roots on their way to the gray matter of the cord. In most cases which come to the post-mortem table the median fasciculi of the posterior columns

or the columns of Goll are likewise sclerosed, but this alteration is is to be looked upon as secondary, as a form of ascending degeneration, and probably as wholly disconnected with the symptoms. Almost always sclerosis of the external part of the posterior columns, or columns of Burdach, commences in the lumbar enlargement and advances upward. Ultimately, in old cases, the whole of the posterior columns as high as the medulla oblongata is sclerosed and degenerated, atrophied and hardened. Another part of the lesion of locomotor ataxia is a sclerosis and degenerative change in the posterior roots extending to the ganglia on these roots and even involving them. In uncomplicated cases of locomotor ataxia, the anterior and antero-lateral columns, and the whole of the gray matter of the spinal cord, are healthy. The symptoms of the disease are chiefly of a sensory sort, and there is never any true paralysis in uncomplicated cases. The chief symptom in some respects, the initial symptom in the vast majority of cases, is a peculiar, almost pathognomonic neuralgia, which usually has its seat in the lower extremities alone, and sometimes affects the upper extremities and trunk, very rarely the head. The diagnosis of locomotor ataxia depends so much upon this symptom and its exact appreciation that I need no other apology for stating its characteristics with some fulness.

a. The pains are vagrant; they occur in innumerable spots in the affected parts, so much so that patients who have long had them are unable to enumerate the localities in which they have suffered; or, rather, they can hardly name a region which has escaped.

b. The pains do not occur in the course or distribution of recognized nerve trunks and filaments; they are local pains, and this peculiarity may serve (with *a*) to distinguish between the pain of sclerosis and true neuralgia (sciatica, etc.).

c. The seat of the pain is commonly in an area of skin

varying in size from that of a pea to that of a small hand. In many cases pains are referred to the muscles, to the vicinity of the bones, and even to articulations and viscera.

d. The pains are paroxysmal in a completely irregular manner; they may occur every few moments for hours in one spot, or be altogether wanting for weeks; or at times a single pain in a given region is the signal that the disease is not cured. It seems probable that the atmospheric disturbance (low barometer) which precedes a storm causes an increase in this symptom, or even calls it forth.

e. The pains are sudden, and vary in severity from the sensation caused by the penetration of a small knife-blade to that we may imagine to result from tearing through the tissues with a hook or large knife; or, the sensation is like an electric pain in suddenness. Perhaps most of the suffering in such cases is in the shape of stabbing pains in an ovoid or round area of skin (foot, thigh, arm, or shin) repeated every few seconds for hours, or even a whole day. The pain is often such as to make the strongest-willed man writhe and shriek. The seat of pain becomes hyperalgesic —*i. e.*, painful to the lightest touch; yet firm pressure may give relief. From their suddenness and electric character the pains of locomotor ataxia are often called fulgurating, or terebrating.

The *rationale* or physiology of these characteristic pains is found in the morbid anatomy. As described above, there is in this disease a sclerosis of the connective tissue in the posterior columns of the spinal cord, and chiefly in their lateral portions—*i. e.*, those portions which are traversed by fibres of the posterior roots. Irritation of these sensory nerve fibres is produced by the advancing sclerosis, and probably varies in degree according to changes in the circulation in the diseased spinal cord.

It should be borne in mind that fulgurating pains usually

precede ataxia by three or four years; but this neuralgic period may be shorter (a few weeks) or much longer, perhaps indefinite (in a case of my own, twenty-nine years).

Other sensory symptoms of posterior spinal sclerosis are numbness and anæsthesia. The numbness often goes hand in hand with the pains, and is probably due to the same cause —viz., irritation of the posterior roots and their intra-spinal expansion. It usually affects the feet first, and seems to ascend. When the upper extremities are involved, the numbness first shows itself in the finger tips.

Anasthesia is present in a large majority of cases; probably in all which enter the second period—viz., the ataxic period. It may progress to such an extent as to make the patient perfectly unaware of any excitation applied to the legs (or hands); and to render him ignorant of the positions and existence of the limbs without the aid of sight or touch, the patient "loses his legs in bed." The anæsthesia is caused by the actual destruction or great compression of the sensory fibres already referred to.

Destruction of the myeline and pressure on the axis cylinders by the sclerosed interfibrillar tissue may also explain a singular symptom—viz., the retardation of sensations—*i. e.*, the occurrence of a measurable time (10 to 120 seconds) between the pricking of a part and the acknowledgment of the pain by the patient.

Other symptoms of scelerosis of the posterior columns are motor to all appearances, yet in reality depend upon interference of the disease with the spinal sensory apparatus.

First. Diminished reflexes, pupillary, cutaneous, tendinous and visceral. For example, in many cases the pupils are small (may be unequal), and do not appreciably respond to light and shade, though they do change under accommodative efforts.

Again, if we tap the ligamentum patellæ in a patient

suffering from fulgurating pains, or in one who has entered the ataxic stage, we observe that the quadriceps extensor femoris does not contract and cause a movement of the leg (the knee being semi-bent) as in health. This is known technically as absence of the patellar reflex, a new and most important symptom of posterior spinal sclerosis, one which in my experience hardly ever fails, and which deserves to be ranked as at least equal in importance with the fulgurating pains. Reflexes from cutaneous surfaces are also diminished or lost. The visceral reflex actions by which we micturate, defecate, and produce the sexual orgasm are likewise progressively impaired; and thus we find these patients impotent as a rule, and presenting constipation and slow, imperfect micturition as symptoms.

This reduction in reflexes is caused by disease of the intraspinal sensory parts connected with the various organs and parts we test; the arc for reflex actions is impaired in the posterior columns of the spinal cord or in corresponding regions of the spinal tract.

The ataxic movement of the legs (and of the arms in some cases) which are characteristic of the second stage of the disease, appear at a variable period after the beginning of the neuralgic stage (three months to ten years or more) and is essentially characterized by an irregular, asynergic action of the muscular groups which serve to produce a given movement. The legs are jerked forward and outward, and the heel brought forcibly down in the attempt to walk; the fingers and arms oscillate and perform unnecessary excursions in trying to reach a given point, or accomplish a given action. Later, the irregularity of movement is so great that the patient is confined to bed.

We are not yet agreed upon a theory of ataxia, but these hypotheses are deserving of consideration.

a. That interference with sensory intra-spinal tracts dimin-

ishes the muscular tonus (chiefly produced in an unconscious reflex way), and that this atony varying in different muscles gives rise to the inharmonious movements.

b. The anæsthesia affects the muscles as well as the superficial parts, and thus diminution or loss of the "muscular sense" is caused and the patients can no longer guide their contractions.

c. The sclerosis of the posterior columns affects other fibres besides common sensory ones—viz., those arciform or longitudinal commissural fibres described by Lockhart Clarke, and which seem to unite, for the purpose of harmonious action, the spinal centres for the various muscles of a group, or of a limb. If these commissural fibres be destroyed by sclerosis, we obtain ill-combined, asynergic muscular movements in the attempt to step.

There are many other symptoms in the course of posterior spinal sclerosis, but those just analyzed are the characteristic ones—the ones which are logically related to the lesion.

Under the head of diseases of the æsthesodic region we must also place the lesion of the posterior columns known as ascending degeneration. This lesion is limited to the posterior median columns, or columns of Goll, and diminishes in extent the higher we examine the spinal cord. We find this ascending degeneration above a spot in the spinal cord where it is compressed, or destroyed, and it is also met with in progressive locomotor ataxia. Prof. Charcot and others teach that this extension of sclerosis from the columns of Burdach to the columns of Goll is a non-essential feature of locomotor ataxia, and is secondary. At any rate sclerosis or degeneration of the posterior median columns alone does not, as far as we know to-day, give rise to any special symptoms; hence I can give you no clinical picture to accompany the pathological statement. We know only that the lesion exists

in a living patient by learning the pathological state of the cord from other symptoms present.

2. Diseases of the kinesodic system, including the anterior gray horns, the anterior columns, the antero-lateral columns, and the postero-lateral columns (or crossed pyramidal fasciculi).

There are, as indicated in the table, several very definite and distinct affections of those parts.

a. Degeneration of ganglion cells in the anterior horns. In this disease the protoplasm of these cells is very slowly transformed into granulo-fatty material, the cell processes are broken off, the nucleus and nucleolus destroyed, and in later stages there remains of the cell only a small mass of granules, or its place in the tissues may be vacant. These changes take place with extreme slowness, and the symptoms are correspondingly gradual in their appearance. For reasons which we do not understand, this granular degeneration may be limited to a few groups of ganglionic cells, or may involve a large number of the cells from the medulla oblongata to the end of the cord. We are not acquainted with the lesion above the medulla, although there is no reason why the nuclei of the fifth, sixth, seventh, fourth and third cranial nerves should not suffer. I wish to lay particular stress upon the slowness of the degeneration because of its harmony with symptoms, and its contrast with allied states.

Degeneration of anterior ganglion cells is externally expressed by muscular atrophy—usually progressive muscular atrophy. This singular disease shows itself often first in peripheral muscles, in hands and feet, in other cases it attacks the shoulder, arm and thigh muscles. Indeed, it may commence in any muscular group. Yet there are certain characteristic peculiarities in this wasting.

1. It is very gradual; fasciculi after fasciculi in the muscles undergo a diminution in size, lose their faradic contractility,

and disappear, while adjacent fasciculi remain normal. There
is no *paralysis* of a muscle, but a partial and gradual death
of its constituents.

2. The atrophy affects, in the vast majority of cases,
symmetrical and homologous parts. For example, both
shoulders may be wasted, or the arms and thighs, or the
forearms and legs. Unilateral muscular wasting is presum-
ably not progressive muscular atrophy.

3. The muscles which are undergoing the early changes
of this wasting are the seat of what are called fibrillary con-
tractions. These are produced by the involuntary rapid
contraction of fasciculi of muscular fibres in a muscle.
Sometimes a patient is covered with them. Some years
ago these fibrillary contractions were held to be patho-
gnomonic, but I can assure you that this is not so, as they
may be observed in lead palsy, in conditions of neurasthenia,
in simple paralysis. Indeed, many years ago, Prof. Schiff,
now of Geneva, showed that muscles separated from their
motor nerves were prone to show fibrillary contractions.

The electrical reactions are diminished, but not altered in
quality; and the reduction or loss of reaction (to faradism)
is in direct proportion to the wasting. In partly-atrophied
muscles some fasciculi look well and contract well, while
their neighbors are thin, and rise but feebly under faradism.

Uncomplicated degeneration of ganglion cells is unaccom-
panied by numbness, anæsthesia, or vesical symptoms,
though aching pains may be present in the affected regions.

b. Inflammation of the anterior gray horns; myelitis
anterior in adults and in children.

Autopsies have shown that the lesion in this disease in-
volves the anterior and central gray matter of the spinal
cord, and that there may also be present, as secondary con-
ditions, atrophy and degeneration of the antero-lateral
columns (not distributed as in regular descending degenera-

tion). The ganglion cells of the affected anterior horns are found in various states of change — swollen, containing vacuoles, filled with granulo-fatty matter, or shrunken and almost destroyed. Besides, the neuraglia round about these cells is always more or less altered. In some cases the lesion might be designated as a diffused central myelitis with destruction of anterior ganglion cells. It need hardly be added that there exists an absolute anatomical relation between the level at which the changes occur in the spinal cord and the distribution of symptoms at the periphery.

The symptoms of myelitis anterior are chiefly motor, very much as in group (*a*); but the morbid process being comparatively or absolutely rapid, they appear in a strikingly acute or sub-acute form.

First. Paralysis.—In some few cases this appears with almost apoplectic rapidity (suggesting hæmorrhage in the cord); in the majority the paralytic phenomena are complete in one or two days. Often we learn from the mother of a child who has myelitis anterior (infantile spinal paralysis) that she put the child to bed well, or simply a little feverish, and that the next morning both legs, or one extremity, or all the limbs were limp and paralyzed. In a minority of cases (adults) the muscular groups in the limbs lose power slowly, and a week or several weeks elapses before the patient is obliged to lie a-bed. It is important to contrast this paralytic loss of power, affecting a whole limb, or a whole muscular group, with the atrophic failure of power, or the fascicular atrophy described under the head of " Progressive Muscular Atrophy."

Second. Atrophy of muscles.—The palsied muscles in myelitis anterior always undergo, in the course of the first four or five weeks, marked and even extreme atrophy. This wasting, even in the most sub-acute forms of myelitis anterior, is rapid as compared with the most rapid forms of

progressive muscular atrophy. Besides, in the former disease, the atrophy, like the paralysis, always affects a whole muscle, or a muscular group, or the muscular apparatus of one or more limbs, *en masse*, and is not, as in the latter affection, fascicular in distribution. Fibrillary contractions in wasting muscles are common in progressive muscular atrophy, and very rare in myelitis anterior.

Third. Remarkably distinct evidences of the degeneration reaction to electricity are obtained from the second to the tenth week. The nerve-trunks supplying the paralyzed muscular groups lose their excitability to faradism and galvanism, and these wasting muscles react only to galvanism, and that their reaction formula is altered from the normal; in general terms, we may say that An c c = Ka c c, or even An c c > Ka c c; and all contractions are slow and wavelike.

Fourth. The distribution of the paralysis is important for diagnosis and prognosis. In myelitis anterior the bladder and rectum are never paralyzed, and it is exceedingly rare to observe paralysis of the respiratory muscles, and of those which serve for deglutition. On the other hand, muscles supplied by the cranial nerves may be paralyzed.

Fifth. An important symptom not usually referred to is abolition of reflexes in the paralyzed parts, especially the tendon reflexes. For example, if the muscles of the thigh (quadriceps extensor femoris) be paralyzed, no patellar tendon reflex can be obtained, just as in posterior spinal sclerosis. The mechanism is in both affections the same, but in either case different portions of the spinal arc serving for reflexes are injured; in posterior spinal sclerosis the centripetal (sensory) portion of the arc is destroyed; in myelitis the anterior (motor) portion. At an early stage of myelitis anterior, and in cases (infantile) where much fat serves to obscure atrophy, this negative symptom acquires great value.

Sixth. Sensory symptoms are very slightly developed in myelitis anterior. In some cases a degree of numbness or other paræsthesia is experienced in the paralyzed limb for a few hours or days. In others severe neural pains are experienced. I style these neural because they apparently are in the course of nerve trunks or large branches, and are not in spots or patches like the fulgurating pains of posterior spinal sclerosis. Although the suffering in some few cases of myelitis anterior may be very severe, yet it is only careless observers who could mistake these pains for those of locomotor ataxia. Anæsthesia is never present in myelitis anterior. When it is observed in any marked degree the case should be designated as diffused central myelitis.

Seventh. Fever is present in a majority of cases in children and adults. Other peculiar symptoms are vomiting, œdema of the extremities, hyperæsthesia of affected parts, delirium. These rare symptoms occur in the first stage of the disease. In the atrophic stage the patient's general health is usually good.

c. Sclerosis of the lateral columns has been recently described by Erb (1875) and Charcot (1876) as probably an independent or protopathic lesion of the spinal cord. To the symptom-group characteristic of this lesion, Prof. Charcot has given the name of spasmodic tabes, Prof. Erb that of spastic spinal paralysis. I have proposed the name of tetanoid paralysis or paraplegia, which seems to me most expressive.

The pathological anatomy of this affection is not as yet well established; it is doubtful if the lateral columns generally are sclerosed, or whether the lesion is limited to their posterior positions, as in descending degeneration (*vide infra*). The lesion is always (?) bi-lateral. Another uncertain point is whether the lateral sclerosis is primary or secondary—*i. e.*, due to a focus of disease centrally placed above the begin-

ning of the sclerosis. It seems to me that the weight of evidence is in favor of the latter view.

The symptoms of tetanoid paraplegia consists in slowly increasing paresis in the legs (and arms rarely) with tendency to contracture and increase in all reflexes. The loss of power is exceedingly slow. The patellar tendon reflex is increased very early in the disease, and becomes greatly exaggerated. The skin of the feet form the starting point of reflex muscular contractions when the patient is awake. Except at the close of the disease the affected limbs are relaxed in sleep. As a result of this increase of reflex action, there is a peculiar attitude and gait. The legs tend to cross one another, or actually do so from over-action of the adductors. The heel is drawn up, or at any rate raised, by over-action of the gastrocnemii and solei; the legs in stepping are stiffened, their muscles sensibly hardened. The extremities are in a tetanoid condition. In later stages, in bed-ridden patients, the legs may be fixed in semi-flexion and adduction. These symptoms indicate clearly that the proper spinal activity, its reflex and automatic activity, is increased.

Another motor disturbance, due to increased reflex action, is precipitate micturition and defecation. The patient must hurry, as from increased reflex action the bladder and rectum are apt to contract suddenly and involuntarily upon their contents. We have here an example of incontinence through spasm—a condition to be carefully distinguished from paralytic incontinence. The muscles in parts affected with tetanoid paralysis retain their volume, nutrition, and normal electrical reactions.

Sensory symptoms are practically wanting in this disease; there is no anæsthesia, and seldom numbness.

If there were time I should like to draw a contrasting picture between this condition and sclerosis of the

posterior columns; you can, however, easily do it for yourselves.

In little children we not very rarely meet with the symptoms of tetanoid paralysis involving the legs alone, or all the limbs. The child cannot stand or walk because of the spasmodic condition of the legs and the apparent absence of cerebral stimulus; no anæsthesia is present. In some cases we find microcephaly and idiocy conjoined. Prof. Erb was the first to describe these infantile forms (1877), and he is disposed to think that pathological researches will show as lesions, sclerosis or want of proper development in the postero-lateral colums. I incline to the view that in these tetanized children the entire motor tract, from its cortical (cerebral) starting point to its distribution in the spinal cord, is more or less incompletely developed. The operation of circumcision is still gravely advocated as a cure for infantile tetanoid paralysis, but we have no recent and uncontradicted statements of cures; besides, in my experience, Jewish children fully circumcised from birth frequently come to my clinic with these symptoms.

A combination of *b* and *c* is recognized under the name of amyotrophic lateral sclerosis (Charcot).

In this affection the lesions proper to *b* and *c* respectively are found in the spinal cord—viz., destruction of the anterior ganglion cells, and sclerosis (degeneration, probably) of the postero-lateral (or crossed pyramidal) columns.

The symptoms correspond. We have a paralysis with atrophy in the upper and lower extremities, followed by contractions, with increased reflex in the non-atrophied and non-paralyzed muscles.

d. Descending degeneration: *α.* Of spinal origin. This takes place when the physiological continuity of the spinal cord is interrupted. For example, after division of the cord with a knife, after great pressure from a tumor, or from a

displaced vertebra, or from a focus of transversely localized myelitis, we observe these changes. They differ completely above and below the seat of pressure or of section. Above the lesion we find an alteration almost limited to the posterior median columns (columns of Goll) extending indefinitely upward toward the apex of the fourth ventricle. The degenerated tissue appears in transverse sections as a wedgelike area with its base resting on the pia mater and its apex not quite reaching the posterior commissure (see Fig. 1). Careful examination often shows on each side of the cord another tract of altered nerve tissue—viz., a portion of the postero-lateral columns lying posteriorly to the crossed pyramidal column, and extending to the pia mater and the posterior gray horn—the so-called ascending cerebellar fasciculus (Flechsig).

Below the lesion dividing the spinal cord the degenerations are found chiefly in two locations. First, in the crossed pyramidal columns (see fig. 1); and, second, in the anterior columns, or, physiologically, the direct pyramidal columns. In these columns the descending degeneration extends to the lowermost portion of the cord. A more complete description of these columns will be given in the next section.

The symptoms of the ascending degenerative changes, if there be any, are now unknown. As regards descending changes, they very probably are expressed by a more or less defined tetanoid state of the paralyzed limbs. In some cases this is well marked, and constitutes the spinal epilepsy of Brown-Séquard, or may resemble the symptom-group (*c*) attributed to the primary (?) lateral sclerosis. No sensory symptoms accompany either ascending or descending degeneration.

β. Descending degeneration of cerebral origin :

In this condition the foci of disease are found above the decussation of the anterior pyramids; by far the greater

number within the cerebral hemispheres. The nature of the lesion is, of course, immaterial, providing it be a desrtuctive one. Such, for examples, are clots in the motor regions of the brain (more especially involving the anterior two-thirds of the internal capsule), patches of softening involving the motor convolutions or tumors in similar locations. Usually such a lesion is uni-lateral, but once in a while bi-lateral lesions exist, and we must be prepared for the existence of bi-lateral symptoms.

From the focus of disease in these motor convolutions, or in the associated fasciculi of the corona radiata, the internal capsule (directly or indirectly involved), in general terms from the central part of the motor tract described to you by Dr. Gray, we can trace the degenerative changes with the naked eye (after six or eight weeks) to near the end of the spinal cord, through certain well-known columns or fasciculi of the cerebro-spinal apparatus. Let us take an example : A patch of softening involving the motor convolutions bounding the fissure of Rolando in the right hemisphere, now known to be the source of direct innervation for the left upper and lower extremities. Beneath the patch there is an altered bundle of fibres of the corona radiata, extending downward in the anterior (or middle) portion of the internal capsule ; thence into the right crus cerebri, the right inferior half of the pons, the right anterior pyramid of the medulla oblongata. At the base of the brain, in the crus, pons, and medulla the degeneration is superficially evidenced by an atrophy of the parts and cord. Below the medulla oblongata the changes are to a considerable degree invisible to the naked eye, but appear upon transverse sections of the spinal cord. On examining such sections we find changes in different localities in the two halves of the spinal cord. In the immense majority of subjects, the evident, the principal change, or secondary degeneration, is found in the

left posterior lateral column, or, more strictly speaking, the crossed pyramidal column, so called because it is the continuation across the median line of the anterior pyramid which has (partially) decussated at the point known as the pyramidal decussation. The exact location of this bundle (in the transverse section) varies somewhat at different levels in the spinal cord. In the upper cervical region it lies deep in the lateral columns, and as we examine sections made lower down we find it more posteriorly and laterally. It is always in the posterior-lateral column, to use a rough anatomical term, and never quite reaches to the periphery of the cord; it diminishes in size (area) from above downward, and is quite lost to the naked eye in the lower lumbar enlargement. In this crossed pyramidal column (see fig. 1), I repeat, the principal descending degeneration is found. The other, smaller descending lesion, in our supposed case, is found in the right half of the spinal cord—*i. e.,* on the same side as the original disease and the intra-cranial degeneration-paths. This second spinal degeneration involves the anterior column of the cord, strictly speaking—*i. e.,* that fasciculus which lies between the anterior median fissure and the anterior gray horn—the so-called column of Türck (see fig. 1). This column embraces those fibres of the anterior pyramid which have not crossed the median line; hence the better name for this fasciculus is the direct pyramidal column. In our supposed case we find the right anterior column more or less altered throughout the length of the cord.

To sum up: Below the pyramidal decussation the secondary degeneration is found both in the crossed and the direct columns derived from one of the pyramids—the right in our hypothetical case.

A most important law, discovered and abundantly proved by Prof. Flechsig, of Leipzig, is that of *variability in the pyramidal decussation*. He found that in most of his speci-

mens the crossing took place as above described, the great majority of pyramidal fibres from the anterior pyramid crossing the median line and going to form the crossed pyramidal column, while a smaller number remained uncrossed and constituted the direct pyramidal column. But in one of his fœtuses this was reversed—*i. e.*, the direct pyramidal column was much larger than the crossed column. In several instances he found a degree of equality between the two columns derived from a pyramid. He concludes—and I entirely agree with him—that we must look upon the old law of decussation of motor fibres as liable to rare but remarkable variations. One result of such reversal of the law of decussation in a given individual would be to cause the paralysis from a cerebral lesion to appear on the same side of the median line. Although Brown-Séquard has written on the subject of paralysis from cerebral disease since the publication of Flechsig's great work (1876), he ignores these facts, which are alone sufficient to maintain the accepted hypothesis of cerebral motor action, and also to completely refute his iconoclastic theories, since, if we admit that sometimes the pyramidal distribution is chiefly direct, we must deny the value of Brown-Séquard's three hundred cases of paralysis on the same side as the cerebral lesion. As long ago as January, 1878, I made use of this argument, which to my mind is convincing.

To return to the subject of descending degeneration:

Its symptoms consist in increased reflexes (from tendons especially) and contracture, in the wholly or partially paralyzed parts; arm and leg, rarely the face. It is useless to further describe this condition which we all see so often in uncured cases of hemiplegia. I would, however, call your attention to one important fact which I believe I was the first to notice and draw a conclusion from—viz., that during sleep in a warm bed the affected limbs are wholly or par-

tially relaxed, and contract at once upon waking or upon exposure to the cooler air. I need hardly add that the contracture, like the paralysis, in hemiplegia, is greatest in peripheral parts.

The classic theory of the physiology of contracture in hemiplegia is that it is due to the secondary degeneration—*i. e.*, actively caused by the lesion of the postero-lateral column. Seven years ago (see Archives of Scientific and Practical Medicine, vol. I, p. 106, 1873) I rejected this hypothesis, and suggested a different one, which I have since elaborated and taught in my clinical lectures at the College of Physicians and Surgeons, New York. This hypothesis, which I intend shortly to publish in detail, is briefly that the spasm is due, not to direct irritation from the sclerosed (?) tissue in the postero-lateral column, but to the cutting off of the cerebral influence by the primary lesion, and the consequent preponderance of the proper or automatic spinal action—an action which is mainly reflex. This theory explains the phenomena observed in cases of primary spinal diseases with descending degeneration (α) and can be reconciled with results of experiments on animals (increased reflex power of spinal cord after a section high up, Brown-Séquard's; inhibitory power of encephalon on spinal cord, Setchenow).

Want of time will prevent us from considering the focal affections of the spinal cord, classed in the table under section B.